Hypoglycemia and Accidental Hypothermia in an Alcoholic Population

FAITH T. FITZGERALD, MD, Ann Arbor, Michigan

Hypoglycemia is but one of a number of causes of hypothermia, but is important to keep in mind as a possible precipitating or concurrent event even in those cases in which there are other obvious explanations for decreased body temperature (exposure, alcoholism, starvation, sepsis or hypothyroidism). Hypoglycemia may occur in as many as 40 percent of very cold patients, and be clinically unrecognized because symptoms are masked by the hypothermia itself. Although serum glucose levels are depressed, a cold-induced renal tubular glycosuria may occur. Glucose in the urine, therefore, cannot be used as assurance of hyperglycemia in a hypothermic patient. And, although cold protects against serious end organ damage from hypoglycemia by decreasing tissue metabolic need for glucose, a serum specimen should be drawn for glucose determination in all hypothermic patients and a 50 percent glucose solution immediately given intravenously. If this is not done, serum glucose levels may plummet as the patient is rewarmed and begins to shiver.

HYPOTHERMIA DURING INSULIN THERAPY and insulin-independent hypoglycemia has been recognized for many years.¹⁻⁴ The hypothermia of hypoglycemia is a result of intracellular glucopenia in the hypothalamus, probably mediated by an alteration of neurohumoral controls.⁵ Teleologically, it seems a protective *turning down* of body temperature, and therefore of metabolic rate, in the face of inadequate fuel supply.

Hypothermia may also cause hypoglycemia. In cases of prolonged hypothermia⁶ and in starved hypothermic persons,⁷ low serum glucose values are usual. In addition, a cold-induced renal tubu-

lar glycosuria may actually contribute to hypoglycemia by wasting glucose through the kidneys.⁷

Other influences in cases of hypothermia oppose the development of hypoglycemia. Tissue consumption of glucose decreases as temperature falls.⁸⁻¹³ Decreased pancreatic insulin release,¹⁴⁻¹⁷ decreased tissue sensitivity to insulin,¹⁰ liver dysfunction⁸ and cold-induced pancreatitis¹⁸⁻²³ may all contribute to elevation of serum glucose in hypothermic patients.

Excess ingestion of alcohol is a common associate of accidental hypothermia^{18,24-28} and alcohol may induce hypoglycemia.²⁹⁻³¹ The case records of 22 hypothermic, predominantly alcoholic patients were obtained from medical records by selecting the diagnostic code for hypothermia. These records were retrospectively reviewed in

From the Department of Internal Medicine, the University of Michigan Medical School, Ann Arbor.

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Reprint requests to: Faith T. Fitzgerald, MD, Director, Inpatient Programs, Dept. of Internal Medicine, University of Michigan Hospital, Ann Arbor, MI 48109

order to study the relationship of hypothermia to glucose levels.

Patients and Methods

The 22 patients (19 men and 3 women) all were admitted by way of the emergency room of the San Francisco General Hospital between 1975 and 1977. All had rectal temperatures no higher than 34.4°C (94°F). All but two were alcoholic. Only one was younger than 50 years. Of the 22 patients, 12 were found indoors, in apartments or hotel rooms.

In all 22 a serum specimen for glucose determination was drawn on entry, before parenteral administration of glucose. The highest serum glucose value was 314 mg per dl in a patient with chronic pancreatitis (case 2). The lowest was 13 mg per dl in an alcoholic patient (case 11). In nine patients (41 percent) serum glucose levels were lower than 50 mg and in five (23 percent) they were higher than 150 mg per dl. Analysis of

urine specimens showed three patients to be glycosuric. Of these, two had concurrent serum glucose levels of less than 80 mg per dl. In one patient the urine reaction for glucose was 2+, with a serum glucose value of 18 mg per dl. In the other the urine reaction for glucose was 1+, with a serum glucose level of 76 mg per dl (cases 15 and 6).

Notes in the case records referring to nutritional status were made in only two cases: One man was "debilitated," another "thin." In six patients, however, there were serum albumin values of less than 2.5 grams per dl; it would be difficult to consider a patient with this level of albumin well nourished. Five of these six were hypoglycemic.

In two of the patients a clinical diagnosis of alcoholic pancreatitis was made during the admission for hypothermia. One of these was hyperglycemic (case 1) and one hypoglycemic (case 10). In one patient with insulin-requiring diabetes

		TABLE	1.—Serum	n and Urine Glucose in Twenty-two Hypothern	nic Patie	nts	
Case	Age	Sex	Temp. F°	Diagnosis	Serum Glucose mg/dl	Urine Glucose (Dipstick)	Outcome
1	51	M	84	ETOH, seizure, pancreatitis, GI bleeding, profound folate deficiency (hematocrit=10%	157	(-)	Died
2	39	M	87.6	ETOH, seizure, chronic pancreatitis, sepsis, shock, renal failure, arrest	317	2+	Died
3	62	M	80	ETOH, urinary tract infection, hypoxia	85	(-)	Alive
4	55	M	82	ETOH, parietal fracture, epidural hematoma, SIADH	110	(-)	Alive
5	. 80	M	93	ETOH, ketosis	200	(-)	Alive
6	. 58	M	85	ETOH, cirrhosis, ascites, liver failure, DIC, cardiopulmonary arrest	76	1+	Died
7	. 85	M	95	Cancer pancreas, diabetes, respiratory arrest	25	(-)	Died
8		M	86	ETOH, Gram-negative sepsis, hypothyroidism	26	(-)	Alive
9	70	M	92	ETOH, hypertension	42	(-)	Alive
10	. 53	M	92	ETOH, cirrhosis, anemia, GI bleeding, pancreatitis, cardiac arrest	37	(-)	Alive
11	65	M	83	ETOH, Gram-negative sepsis, ATN, pulmonary edema, GI bleeding	13	(-)	Died
12	. 74	M	88	ETOH, cancer prostate, dementia, GI bleeding	102	(-)	Alive
13	67	M	85	ETOH, Gram-negative sepsis, hypotension	47	(-)	Alive
14	55	M	90	Myxedema, pneumonia	175	(-)	Alive
15	70	F	75	ETOH, ATN, pneumonia	18	2+	Alive
16	68	M	92	ETOH, cirrhosis, hepatoma	112	(-)	Died
17	50+	F	87	ETOH, cirrhosis, ascites	90	(-)	Alive
18	68	M	84	ETOH, dementia, hypothyroidism	40	(-)	Alive
19		F	89	ETOH, Gram-negative pneumonia	95	(-)	Alive
20		M	91	ETOH, Wernicke-Korsakoff encephalopathy	175	(-)	Alive
21		M	84	ETOH, sepsis, pneumonia	128	(-)	Alive
22	59	M	86	ETOH, Gram-negative sepsis, ATN	35	(-)	Died

ATN = acute tubular necrosis GI = gastrointestinal

ETOH = alcohol use or alcoholism

DIC = disseminated intravascular coagulation SIADH=syndrome of inappropriate antidiuretic hormone

and pancreatic carcinoma the serum glucose value was 25 mg per dl (case 7). No other patient was known to have diabetes. Of four patients with cirrhosis of the liver listed as an admission diagnosis, only one was hypoglycemic (case 10), and he had concurrent pancreatitis.

Of the nine hypoglycemic patients, four died (44 percent). There were also four deaths among the remaining 13 patients with serum glucose levels greater than 50 mg per dl.

A listing of admission data, diagnoses, glucose values and outcome is given in Table 1.

Discussion

As may be seen in Table 1, the multiplicity of problems seen in this small series of patients prohibits making valid inferences about the influence of hypoglycemia on morbidity or mortality. It was supposed that some of our hypothermic patients might be hypoglycemic, drawn as they were from a population predisposed to hypoglycemia: malnourished^{32,33} alcoholic persons.²⁹⁻³¹ But the fact that 41 percent of our patients were hypoglycemic made this a surprisingly common finding in alcoholic accidental hypothermia. In a study by Weyman and his colleagues²⁴ of 39 cases of hypothermia—82 percent of which had alcohol listed as a predisposing factor—in only one was the serum glucose level below 50 mg per dl. In our series there was no reliable relationship between depth of hypothermia and serum glucose levels.

In light of these data, all hypothermic patients, unless serum glucose values are known to be normal or high, should receive 50 percent glucose solution intravenously immediately after a serum specimen has been drawn for glucose measurement. Because of hypothermic renal tubular glycosuria, the presence of glucose in the urine of cold patients is no guarantee of elevated levels of serum glucose, and may indeed be contributing to the hypoglycemia of hypothermia.

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